Effect of Salt, Smoke Compound, and Temperature on the Survival of *Listeria monocytogenes* in Salmon during Simulated Smoking Processes

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ABSTRACT: The objectives of this study were to examine and develop a model to describe the survival of *Listeria monocytogenes* in salmon as affected by salt, smoke compound (phenol), and smoking process temperature. Cooked minced salmon containing selected levels of salt (0%, 2%, 4%, and 6%) and smoke compound (0, 5, 10, and 15 ppm phenol) were inoculated with a 6-strain mixture of *L. monocytogenes* to an inoculum level of 6.0 $\log_{10}$ CFU/g. The populations of *L. monocytogenes* in salmon during processing at 40, 45, 50, and 55 °C that simulated cold- and hot-smoking process temperatures were determined, and the effects of salt, phenol, and temperature on the survival of *L. monocytogenes* in salmon were analyzed and described with an exponential regression. At 40 °C, the populations of *L. monocytogenes* in salmon decreased slightly with inactivation rates of <0.01 $\log_{10}$ CFU/h, and at 45, 50, and 55 °C, the inactivation rates were 0.01 to 0.03, 0.15 to 0.30, and 2.8 to 3.5 $\log_{10}$ CFU/h, respectively. An exponential regression model was developed and was shown to closely describe the inactivation rates of *L. monocytogenes* as affected by the individual and combined effects of salt, phenol, and smoking process temperature. Temperature was the main effector in inactivating *L. monocytogenes* while salt and phenol contributed additional inactivation effects. This study demonstrated the inactivation effects of salt, smoke compound, and temperature on *L. monocytogenes* in salmon under a smoking process. The data and model can be used by manufacturers of smoked seafood to select concentrations of salt and smoke compound and alternative smoking process temperatures at 40 to 55 °C to minimize the presence of *L. monocytogenes* in smoked seafood.

Keywords: inactivation, *Listeria monocytogenes*, salt, smoke compound, smoked salmon, temperature

Introduction

Smoked salmon is a ready-to-eat (RTE) product. If not processed and handled properly, smoked salmon can be contaminated with *Listeria monocytogenes*. *Listeria monocytogenes* is a human pathogen that causes listeriosis and, with the ability to grow at low temperatures, it is a pathogen of concern in refrigerated RTE foods. The prevalence of *L. monocytogenes* in cold-smoked salmon or smoked fish has been reported at 10% (Embarek 1994), 9.2% to 13.8% (Cortesi and others 1997), 34% to 43% (Jorgensen and Huss 1998), 7.3% (Norton and others 2001), 13% (Nakamura and others 2004), 4.3% (Gombas and others 2003), and 10.3% (Beaufort and others 2007). The levels of contamination were generally low at <10 CFU/g and seldom exceeded 100 CFU/g (Cortesi and others 1997; Gombas and others 2003; Beaufort and others 2007). Smoked salmon and smoked fish contaminated with *L. monocytogenes* have been implicated in outbreaks of listeriosis. One outbreak that resulted in 9 cases of listeriosis and 2 deaths in Sweden was linked to eating smoked rainbow trout or salmon (Ericsson and others 1997). Risk assessments indicated that consumption of smoked salmon contaminated with *L. monocytogenes* may cause an elevated risk of listeriosis (FDA/USDA/CDC 2003; FAO/WHO 2004). Smoked salmon contains approximately 65% to 78% water, 2% to 8% water-phase salt, and 2 to 15 ppm phenol, with a pH of 5.9 to 6.3 and a_w of 0.95 to 0.98. The salt and moisture contents and pH of smoked salmon and the prevailing storage temperatures for smoked salmon were supportive for the growth of *L. monocytogenes* (Guyer and Jemmi 1991; Dalgaard and Jorgensen 1998; Yoon and others 2004). Smoked salmon is produced by salting, smoking, trimming, or slicing the fish, and then vacuum-packaging the final product. Salting is commonly done by applying 2% to 4% dry salt to the fish or by submersion or injection of the fish with a 70% to 80% brine solution. After salting, the fish or fillets are smoked in a smoke chamber at 20 to 30 °C for 2 to 4 d (cold smoking) or at >60 °C for 6 to 10 h (hot smoking). Phenolic compounds, such as guaiacol, eugenol, and syrigol are major components in smoke compound (Duffes 1999). The compounds, from wood smoke or liquid smoke, have been reported to inhibit the growth of *L. monocytogenes* in smoked salmon (Vitt and others 2001; Gimenez and Dalgaard 2004; Cornu and others 2006). The reported inhibitory effects of smoked compound against *L. monocytogenes* varied due to different types of smoke compound, phenolic composition in smoke compound, and sensitivity of the test strains of *L. monocytogenes* to smoke compound used in these studies (Sunen 1998; Cornu and others 2006). The effect of salt and phenol in decreasing the viability of *L. monocytogenes* in smoked seafood at the prevailing storage temperatures for product distribution has been reported. It is generally recognized that higher concentrations of

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doi: 10.1111/j.1750-3841.2009.01377.x

THE Society for Food Science & Technology
Journal of Food Science
A Publication of the Institute of Food Technologists

salt or phenol increase the growth inhibitory effect against *L. monocytogenes*. The fabrication processes such as trimming, salting, and packaging for smoked seafood are not effective ways of reducing *L. monocytogenes* on smoked salmon. Smoking at adequately high temperatures is a step in the processes that is capable of reducing *L. monocytogenes* contamination on the fish. The temperatures for cold smoking (20 to 30 °C) process are not sufficient to inactivate *L. monocytogenes*. A study that examined the survival of *L. monocytogenes* in smoked salmon during its manufacturing process showed that the populations of inoculated *L. monocytogenes* remained the same after marinating and smoking at 26 to 30 °C (Guyer and Jemmi 1991). While temperatures for hot smoking (>60 °C) can inactivate vegetative microorganisms, the process produces a finished product with different sensory quality to that of cold-smoked processed salmon. The objectives of this study were to examine and model the survival of *L. monocytogenes* as affected by salt, smoke compound (phenol), and temperature (40 to 55 °C) between the cold- and hot-smoking temperatures.

Materials and Methods

Listeria monocytogenes and inoculum preparation

Six strains of *L. monocytogenes* (NFP7459; serotype 3b, NFP7533; serotype 4b, NFP7554; serotype 1/2b, NFP7712; serotype 1/2a, NFP7735; serotype 1/2a, and NFP7779; serotype 2/1a) from the Microbial Food Safety Research Unit, Eastern Regional Research Center, Agricultural Research Service, U.S. Dept. of Agriculture, were used in this study. Each strain was transferred from a −80 °C stock culture into 10 mL of brain heart infusion (BHI) broth (Difco, Becton, Dickinson and Co., Sparks, Md., U.S.A.) and incubated at 37 °C for 6 h. A loopful of cell suspension of each strain was then transferred to fresh 10 mL BHI broth and incubated at 37 °C for 24 h to reach the beginning of the stationary phase. One milliliter cell suspensions from each strain were mixed together, and the mixture was diluted with sterile 0.1% peptone water (PW) to obtain inocula with *L. monocytogenes* counts of 10^8 CFU/mL.

Table 1 — Means of inactivation rate (standard deviation) of *L. monocytogenes* in salmon processed at 40, 45, 50, and 55 °C.

Treatment	Salt (%)	Phenol (ppm)	Temperature 40 °C	45 °C	50 °C	55 °C
1	0	0	0.002 (0.001)	0.010 (0.001)	0.149 (0.001)	2.81 (0.01)
2	0	5	0.007 (0.001)	0.012 (0.001)	0.145 (0.007)	2.78 (0.11)
3	0	10	0.006 (0.003)	0.010 (0.001)	0.146 (0.003)	2.90 (0.08)
4	0	15	0.005 (0.001)	0.015 (0.001)	0.152 (0.001)	2.82 (0.03)
5	2	0	0.004 (0.001)	0.023 (0.005)	0.154 (0.005)	2.80 (0.18)
6	2	5	0.003 (0.002)	0.025 (0.001)	0.152 (0.008)	2.74 (0.04)
7	2	10	0.004 (0.001)	0.029 (0.005)	0.181 (0.016)	2.95 (0.01)
8	2	15	0.008 (0.002)	0.031 (0.006)	0.167 (0.004)	2.95 (0.05)
9	4	0	0.004 (0.001)	0.023 (0.001)	0.166 (0.008)	2.95 (0.01)
10	4	5	0.005 (0.002)	0.025 (0.001)	0.176 (0.002)	2.89 (0.15)
11	4	10	0.007 (0.006)	0.031 (0.006)	0.187 (0.011)	3.19 (0.09)
12	4	15	0.007 (0.002)	0.029 (0.001)	0.196 (0.032)	3.14 (0.01)
13	6	0	0.006 (0.002)	0.024 (0.001)	0.191 (0.007)	3.03 (0.02)
14	6	5	0.006 (0.001)	0.026 (0.001)	0.169 (0.010)	2.95 (0.05)
15	6	10	0.007 (0.001)	0.029 (0.001)	0.240 (0.029)	3.20 (0.22)
16	6	15	0.010 (0.001)	0.029 (0.002)	0.304 (0.029)	3.51 (0.17)

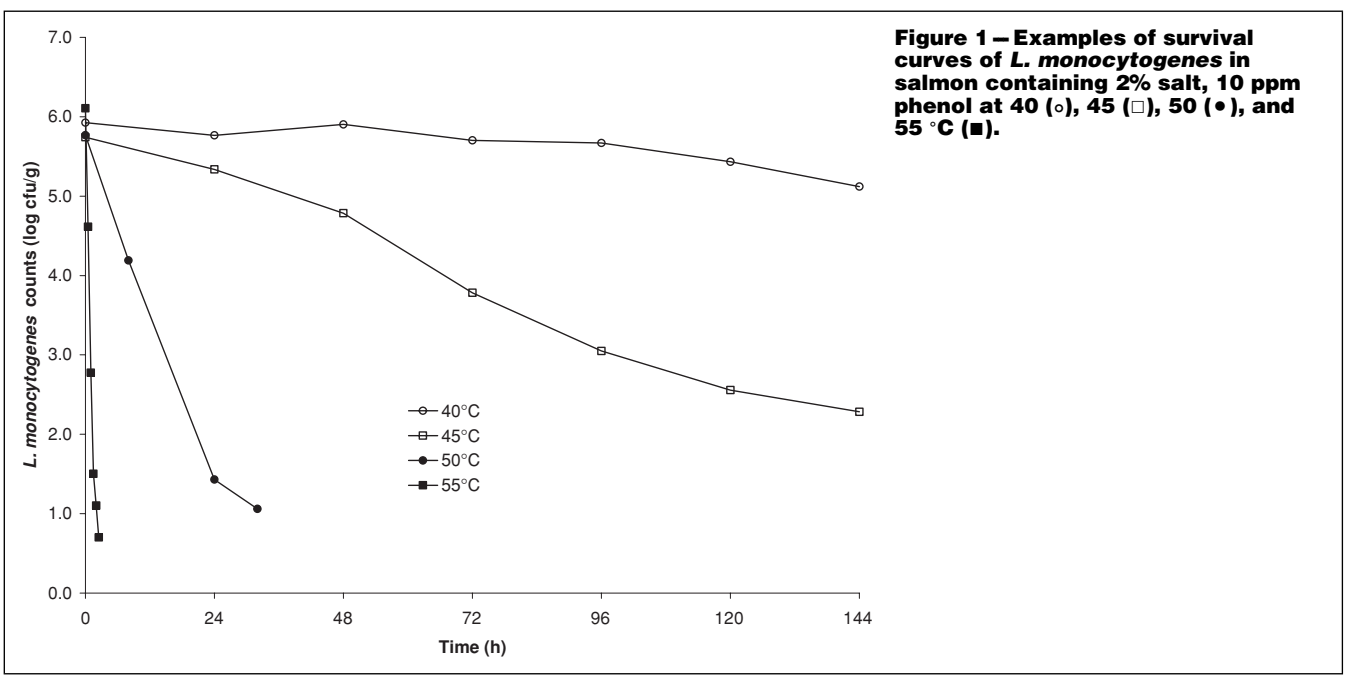

Figure 1 — Examples of survival curves of *L. monocytogenes* in salmon containing 2% salt, 10 ppm phenol at 40 (○), 45 (□), 50 (●), and 55 °C (■).

Sample preparation and smoking process

To ensure the desired amounts and even distribution of salt and smoke compound in salmon samples, a puree of cooked salmon with added salt and liquid smoke was used in this study. Raw Atlantic salmon fillets (15.8% fat) were obtained from a local retail store. Fillets were cut into pieces, placed in stomacher bags (Spiral Biotech, Inc., Norwood, Mass., U.S.A.), and cooked in a water bath to a product temperature of 63 °C and held for 30 min to inactivate the native microflora. Cooked salmon were flaked in the bags by using a sterile spoon. Salmon puree of 60 g were placed in 237-mL sterile polypropylene containers (Fisher Scientific, Pittsburgh, Pa., U.S.A.) and mixed with salt and liquid smoke (Charsol-PN-9, Red Arrow Products Co., Manitowoc, Wis., U.S.A.). The concentrations of salt in the salmon puree were 0%, 2%, 4%, or 6%, and the concentrations of liquid smoke were 0, 0.06, 0.12, or 0.18% (0, 5, 10, or 15 ppm phenol). The samples were inoculated with 0.05 mL *L. monocytogenes* inoculum to achieve an inoculum level of 10^6 CFU/g. The containers were sealed with lids and stored at 40 and 45 °C for up to 144 h, at 50 °C for up to 72 h, and at 55 °C for up to 6 h in incubators (Fisher Scientific, Pittsburgh, Pa., U.S.A.). A complete factorial design ($4 \times 4 \times 4$) as shown in Table 1 was used to examine the effect of salt, smoke compound, and temperature on the survival of *L. monocytogenes* during the simulated smoking processes at 40 to 55 °C. The pH values of the samples, measured by using a Daigger 5500 pH meter (A. Daigger and Co. Inc., Vernon Hills, Ill., U.S.A.), were 6.3 to 6.6, and the a_w, measured by using an AquaLab CX-2 water activity meter (Decagon Devices, Inc., Pullman, Wash., U.S.A.), were 0.95 to 0.98. The experiment was performed in 2 separate trials with 2 samples prepared for each sampling time in each trial.

Enumeration of *L. monocytogenes*

At appropriate sampling intervals (24 h for 40 and 45 °C, 12 to 18 h for 50 °C, and 0.5 h for 55 °C), 2 samples from each treatment were enumerated for *L. monocytogenes* counts. Three grams of sample were placed into a 100-mL filter stomacher bag (Spiral Biotech Inc.), diluted ten-fold with sterile 0.1% PW, and stomached for 2 min in a BagMixer 400 Stomacher. Additional dilutions were prepared with sterile 0.1% PW. From appropriate dilutions, 1 mL or duplicate 0.1 mL aliquots were spread plated onto 3 or 2 tryptic soy agar (Difco) plates, and the plates were incubated at 37 °C for 48 h before typical colonies of *L. monocytogenes* were counted.

Inactivation rates and effect analysis

Mean *L. monocytogenes* counts ($\log_{10}$ CFU/g) from each sampling time during storage were plotted versus time (h) to obtain *L. monocytogenes* survival curves for each treatment. The inactivation rates (slopes of the survival curves, $\log_{10}$ CFU/h) of *L. monocytogenes* were estimated using the linear regression of SAS 9.1 for Windows (SAS Inst. Inc., Cary, N.C., U.S.A.). Inactivation rates for each treatment were plotted versus smoking process temperatures and evaluated with linear and exponential regressions to select a model for fitting. An exponential regression model was found to be preferred, and the inactivation rates were transformed into natural logarithm (ln) for model fitting. The ln-transformed inactivation rates were analyzed by the general linear model of SAS 9.1 as a

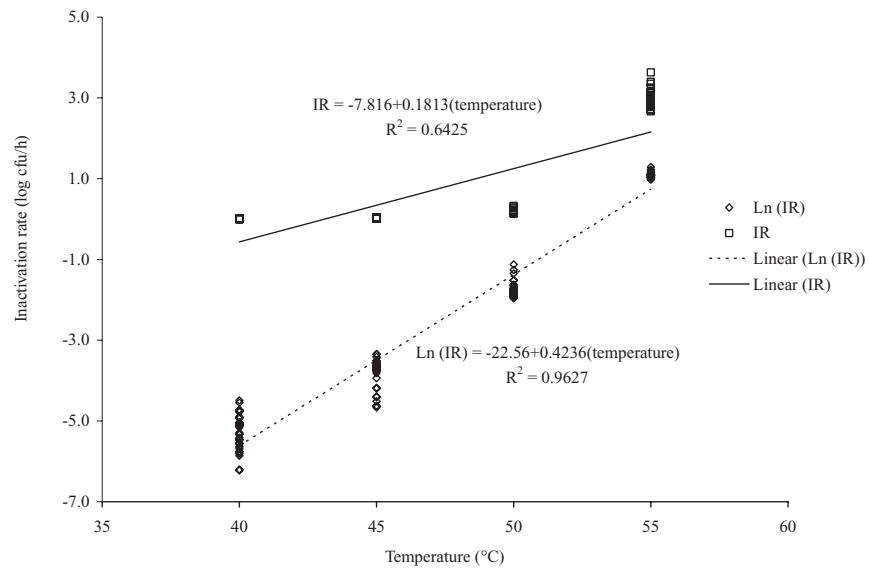

Figure 2—Regressions of inactivation rates in logarithm (◊) and regular (□) forms as a function of temperature.

Table 2—Coefficients for the polynomial regression.

Parameter	Estimate	Standard error	*t* value	Pr > |t|
Intercept	6.4231	1.8993	3.38	0.001
Salt	0.3215	0.0868	3.7	0.0003
Phenol	0.1097	0.0347	3.16	0.002
Temperature	−0.8596	0.0798	−10.77	<.0001
Salt ∗ phenol	0.0012	0.0017	0.7	0.4827
Salt ∗ temperature	−0.0048	0.0017	−2.87	0.0049
Smoke ∗ temperature	−0.0021	0.0007	−3.13	0.0022
Salt ∗ salt	−0.0051	0.0052	−0.98	0.3313
Smoke ∗ smoke	0.0004	0.0008	0.43	0.665
Temperature ∗ temperature	0.0138	0.0008	16.54	<.0001

$R^2 = 0.99$, root mean square error = 0.236.

function of the concentrations of salt (%) and phenol (ppm), temperature, and their interactions using the following quadratic equation:

$$\begin{aligned}\text{Ln(inactivation rate)} = {} & \alpha + \beta 1(\text{salt}) + \beta 2(\text{phenol}) \\ & + \beta 3(\text{temperature}) + \beta 4(\text{salt} * \text{phenol}) \\ & + \beta 5(\text{salt} * \text{temperature}) \\ & + \beta 6(\text{phenol} * \text{temperature}) \\ & + \beta 7(\text{salt})^2 + \beta 8(\text{phenol})^2 \\ & + \beta 9(\text{temperature})^2,\end{aligned}$$

where α is the intercept, and $\beta 1$ to $\beta 9$ are estimated coefficients for each parameter.

Results and Discussion

The initial inoculum of *L. monocytogenes* in all treatments was approximately 6.2 $\log_{10}$ CFU/g. During processing at 40 to 55 °C, the counts of *L. monocytogenes* decreased gradually with greater decreases at higher temperatures. The counts decreased to 5.7 to 4.5 $\log_{10}$ CFU/g at 40 °C after 144 h, 4.7 to 2.0 $\log_{10}$ CFU/g at 45 °C after 144 h, 1.1 to 1.0 $\log_{10}$ CFU/g at 50 °C after 48 h, and <1.0 $\log_{10}$ CFU/g at 55 °C after 2.5 h. Figure 1 shows examples of survival curves of *L. monocytogenes* in samples containing 2% salt and 10 ppm phenol at each smoking temperature. Fitting the *L. monocytogenes* counts verses time (h) with a linear regression showed that the majority of the survival curves (60/64) were considered

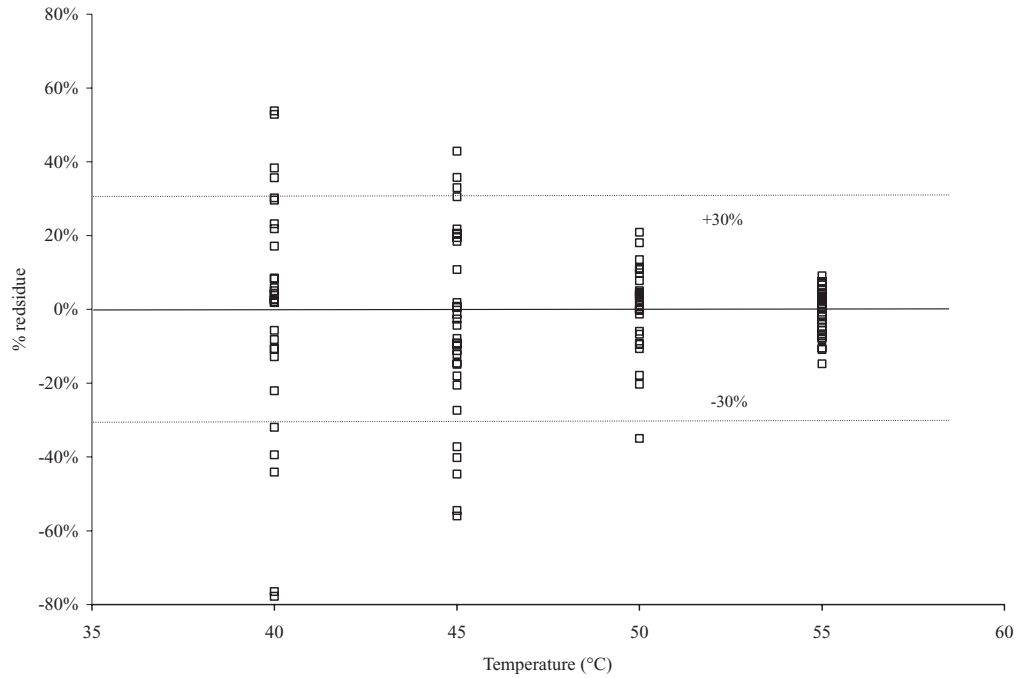

Figure 3—Residue as percentage of observed values at 40, 45, 50, and 55 °C.

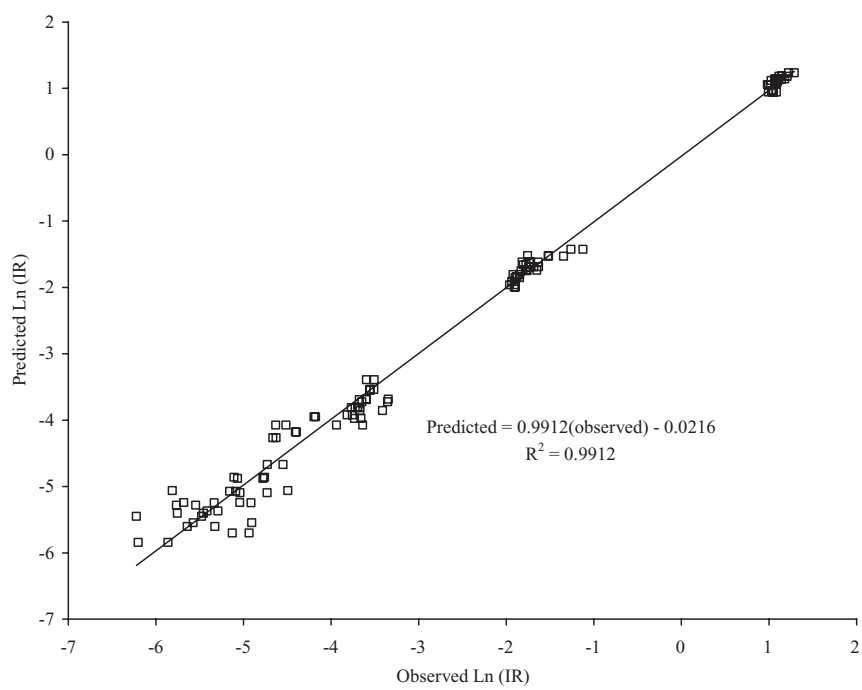

Figure 4—Correlation of predicted IR versus the observed IR.

linear ($R^2 > 0.95$), with 2 survival curves each at both 45 and 50 °C having short shoulder regions prior to the linear reduction. It is not uncommon to observe nonlogarithmic linear inactivation kinetics for microorganisms in heat inactivation studies. Survival curves with shoulder and tail regions have been reported, and nonlinear logistic, modified Gompertz, or sigmoidal equations have been proposed to describe these survival curves (Linton and others 1996; Augustin and others 1998; Ross and others 1998; Huang and Juneja 2001). Since the majority of the survival curves were linear, the inactivation rates (IR, $\log_{10}$ CFU/h) of *L. monocytogenes* in salmon at 40 to 55 °C were the slope of the linear regression of the survival curves. For survival curves with a shoulder region, the IR were calculated from the survival curves that comprised the whole process time. The resulted IR were not significantly different from those calculated from the linear regions ($P > 0.05$). The IR of *L. monocytogenes* in all treatments are presented in Table 1. The IR of *L. monocytogenes* were 0.002 to 0.01, 0.01 to 0.31, 0.15 to 0.30, and 2.7 to 3.5 $\log_{10}$ CFU/h at 40, 45, 50, and 55 °C, respectively. In general, greater IR occurred in samples stored at higher temperatures and/or in samples containing higher concentrations of salt and phenol at the same temperature. The IR increased approximately ten-fold while the temperature increased by 5 °C, indicating that smoking temperature is a main factor affecting inactivation of the pathogen. The narrower ranges of IR in samples of different treatments stored at the same temperature indicated that salt and phenol also contributed to the inactivation effect. This study showed that higher salt or phenol concentrations generally increased the heat inactivation effect on *L. monocytogenes* at 50 and 55 °C. As reported by Juneja and Eblen (1999), an increase in salt concentrations up to 4.5% increased heat resistance of *L. monocytogenes* in simulated beef gravy at 55 to 65 °C. However, they also observed a decreased heat resistance of *L. monocytogenes* when salt was increased to 6%. Results observed from this study indicate that salt concentrations at certain levels, for example, >4%, may adversely affect the heat resistance of *L. monocytogenes*, possibly due to the stress effect of salt. There were studies that reported the heat resistance of *L. monocytogenes* as D values in food matrices at temperatures >50 °C (ICMSF 1997). The D values reported by these studies were transformed to IR to compare to those obtained from this study. At 50 °C, the IR of *L. monocytogenes* were 1.90 $\log_{10}$ CFU/h in reconstituted nonfat dry milk, 0.60 $\log_{10}$ CFU/h in chicken breast meat, 0.34 $\log_{10}$ CFU/h in chicken leg meat, and 0.71 $\log_{10}$ CFU/h in beef. At 54.4 °C, the IR of *L. monocytogenes* were 3.16 to 2.73 and 3.00 $\log_{10}$ CFU/h in roast beef and fermented sausage, respectively. At 55 °C, the IR were 2.86, 4.28, and 4.62 $\log_{10}$ CFU/h in beef, chicken leg, and chicken breast meat, respectively (ICMSF 1997). At 55°C, the predicted IR of *L. monocytogenes* were 3.75, 2.92, 2.69, and 2.94 $\log_{10}$ CFU/h in simulated beef gravy containing 0%, 2%, 4%, and 6% salt, respectively (Juneja and Eblen 1999). At 55°C, the IR of *L. monocytogenes* in minced beef and potato were 7.65 to 9.84 and 5.51 to 6.60 $\log_{10}$ CFU/h, respectively (Walsh and others 2001). The IR of *L. monocytogenes* in bologna batter averaged

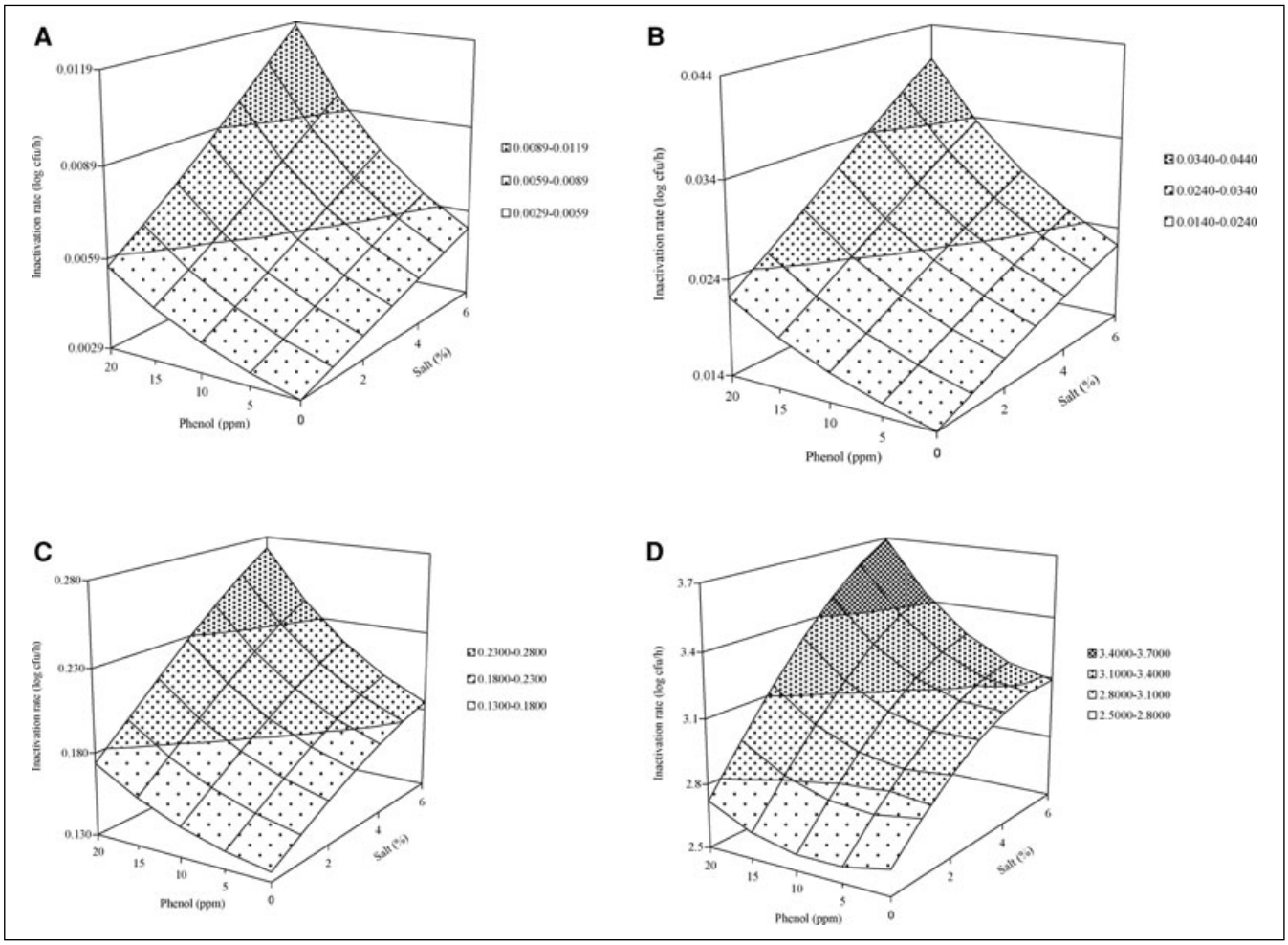

Figure 5—Response surface plots of inactivation rates of *L. monocytogenes* in salmon containing 0 to 5% salt, 0 to 20 ppm phenol at 40 (A), 45 (B), 50 (C), and 55 °C (D).

2.38 and 3.47 $\log_{10}$ CFU/h at 50 and 55 °C, respectively (Sallami and others 2006), whereas the IR were 0.50 and 0.54 $\log_{10}$ CFU/h in 2 bologna samples at 55 °C (Selby and others 2006). In general, the IR of *L. monocytogenes* at 55 °C (2.7 to 3.5 $\log_{10}$ CFU/h) observed in this study were similar to those reported by other studies. At lower temperatures, the IR of *L. monocytogenes* in salmon were generally lower than those in other food matrices. The differences in the heat resistance of *L. monocytogenes* reported by studies are not uncommon (Augustin and others 1998). Studies have indicated that strains of *L. monocytogenes*, growth conditions, and the composition of food matrices used in heat inactivation evaluations affected the heat resistance of *L. monocytogenes* (Juneja and Eblen 1999; Doyle and others 2001; Juneja 2003; Murphy and others 2003).

Figure 2 shows the untransformed- and natural logarithm (ln)-transformed IR compared with the temperature fitting with a

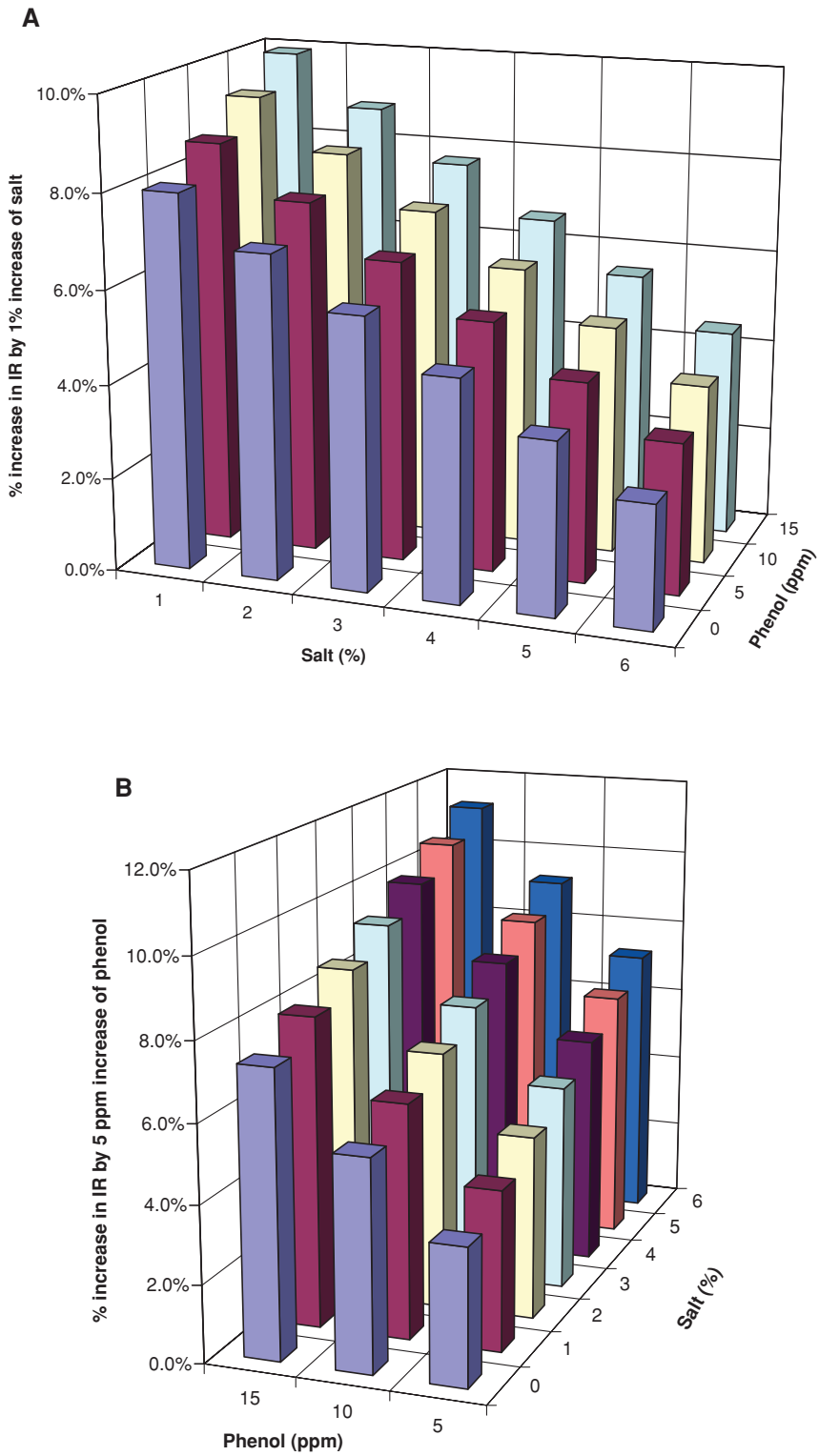

Figure 6—Predicted increases of IR when salt is increased by 1% (A) and phenol is increased by 5 ppm (B) at a smoke processing temperature of 50 °C.

linear regression. The regression coefficient (R^2) was 0.64 for the untransformed IR and was 0.96 for the ln-transformed IR (Figure 2). Based on the R^2, the IR as a function of the main effector, temperature, were better fitted with an exponential regression. Therefore, the ln-transformed IR were fitted with a quadratic equation to examine the effect of salt, phenol, temperature, and their interactions on the IR of *L. monocytogenes* in salmon. The quadratic equation that describes the IR (ln-transformed) as a function of salt, phenol and temperature is

$$\begin{aligned}\text{Ln(IR)} = {} & 6.4231 + 0.3215(\text{salt}) + 0.1097(\text{phenol}) \\ & - 0.8596(\text{temperature}) + 0.0012(\text{salt} \times \text{phenol}) \\ & - 0.0048(\text{salt} \times \text{temperature}) - 0.0021(\text{smoke} \\ & \times \text{temperature}) - 0.0051(\text{salt})^2 - 0.0004\ (\text{phenol})^2 \\ & - 0.0138(\text{temperature})^2.\end{aligned}$$

The fitting parameters, estimated coefficients, and their significance levels are shown in Table 2. The equation was significant ($P < 0.001$) with R^2 of 0.99 and root mean square error of 0.236, indicating a good fit of the IR to the parameters. Salt, phenol, and temperature were significant ($P < 0.05$) in affecting the IR. In addition, there were combined effects from temperature and salt, and temperature and phenol. Predicted IR values were obtained from this equation with the range of concentrations of salt and phenol, and temperature in each treatment and compare to the observed values. The residue values (predicted-observed) in percentage (% residue) of the observed values are shown in Figure 3. Eighty-three percent of the predicted values were within 30% of the observed values. In addition, the correlation between the predicted and observed values was linear with $R^2 = 0.99$ (Figure 4). These results indicate that the equation describes well the IR of *L. monocytogenes* in salmon as affected by salt, phenol, and temperature. The same approach of using environmental factors to describe ln-transformed inactivation rates (as D values) of *L. monocytogenes* has also been reported. Juneja and Eblen (1999) developed a predictive thermal inactivation model for *L. monocytogenes* as affected by temperature, NaCl, and sodium pyrophosphate in beef gravy, and Juneja (2003) developed a predictive model to describe the combined effect of temperature, sodium lactate, and sodium diacetate on the heat resistance of *L. monocytogenes* in beef.

Response surface plots derived from the equation to show the effects of salt, phenol, smoking temperature, and their interactions on the IR of *L. monocytogenes* are shown in Figure 5A–D. In general, the increase of salt and phenol concentrations in salmon and higher temperatures increase the IR of *L. monocytogenes*. The correlations between salt or phenol concentrations and the IR show that the increases of IR are more proportional to the increases of salt or phenol at 40, 45, and 50 °C (Figure 5A–C) than at 55 °C. Examples of the increases of IR in relation to the increases of salt and phenol concentrations at 50 °C are shown in Figure 6. The increases of IR (in percentage) with salt are higher at lower salt concentrations or at higher phenol concentrations. The increases of IR due to the increases of salt are more profound at lower salt concentrations. For example, with 5 ppm phenol, the IR is increased by 8.6% when salt is increased from 0% to 1%, whereas the IR is increased by 3.2% when salt is increased from 5% to 6% (Figure 6A). However, the increases of IR are more profound at higher phenol concentrations. For example, with 2% salt, the IR increase is 4.7% when phenol is increased from 0 to 5 ppm, and the IR increase is 8.6% when phenol is increased from 10 to15 ppm (Figure 6B). Within the temperatures examined, at the same phenol concentration, an increase of 1% salt increases IR by approximately 10% to 12%, 8% to 10%, 5% to 7%, and 3% to 5% at 40, 45, 50, and 55 °C, respectively, with higher increases in samples containing higher salt concentrations. At the same salt concentration, a 5 ppm increase in phenol concentration increases IR by approximately 17% to 21%, 11% to 15%, 5% to 9%, and 0% to 4% at 40, 45, 50, and 55 °C, respectively, with higher increases at higher phenol concentrations. This indicates that the increase of salt or phenol concentrations contribute an added inactivation effect when the temperatures are less stressful to *L. monocytogenes*, that is, at lower temperatures. The added effect of salt or phenol is reduced when the temperature is more stressful to *L. monocytogenes*, that is, at higher temperatures. The added effect of salt and phenol at lower temperatures suggests that salt and phenol are more effective in inactivating *L. monocytogenes* at lower smoking temperatures.

Conclusions

The behavior of *L. monocytogenes* in smoked salmon as affected by salt and smoke compound during storage at refrigerated and abuse temperatures has been extensively studied (Gimenez and Dalgaard 2004; Lebois and others 2004; Cornu and others 2006; Hwang 2007). It is well recognized that higher salt and phenol concentrations inhibit the growth of *L. monocytogenes* in smoked salmon during product distribution. The survivability of *L. monocytogenes* during the smoking process is less well understood. This study demonstrates the survival of *L. monocytogenes* in salmon containing various amounts of salt and phenol at temperatures between the commonly used cold- and hot-smoking temperatures for smoked salmon processes. The data and model developed can be used to assess the effect of salt, phenol and temperature on the survival of *L. monocytogenes* in salmon during alternative smoking process temperatures at 40 to 55 °C. Manufacturers can use this information along with information on the survival of *L. monocytogenes* during storage to reduce levels of *L. monocytogenes* contamination to further enhance the safety of smoked salmon.

Acknowledgments

The author would like to thank Mr. Mark LaSorda of Ursinu Univ., Collegeville, Pa., for performing part of the experiment.

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